

FIGURE NO. 1

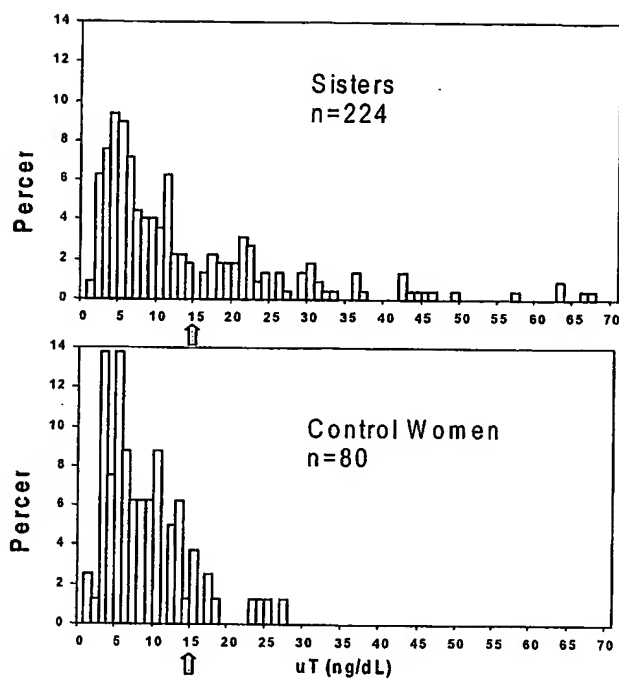


Figure 1. Distribution of uT levels. A uT level of 15ng/dL is 2 SD above the control mean and a value >15ng/dL was used to diagnose hyperandrogenemia. The distribution of uT levels is significantly bimodal ($P < 0.001$) in the sisters, whereas it is not in the controls.

FIGURE NO. 2

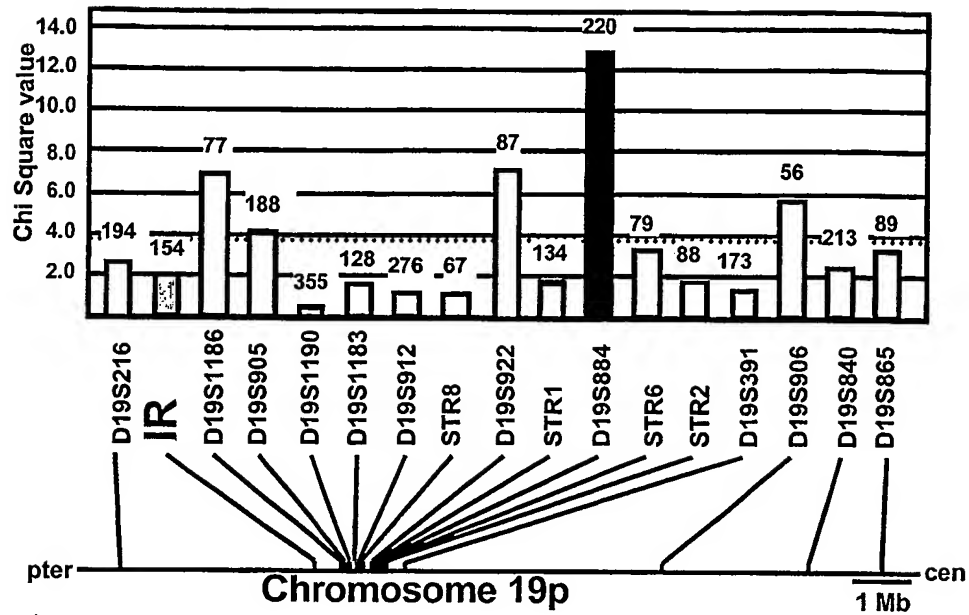


Figure 2. TDT analysis of chromosome 19p, D19S884 $\chi^2=12.95$, $P=3.21 \times 10^{-4}$ with 220 transmissions. Solid bar DS19S884, gray bar IR, dotted line $\chi^2=4$, nominal $P=0.05$.

FIGURE NO. 3

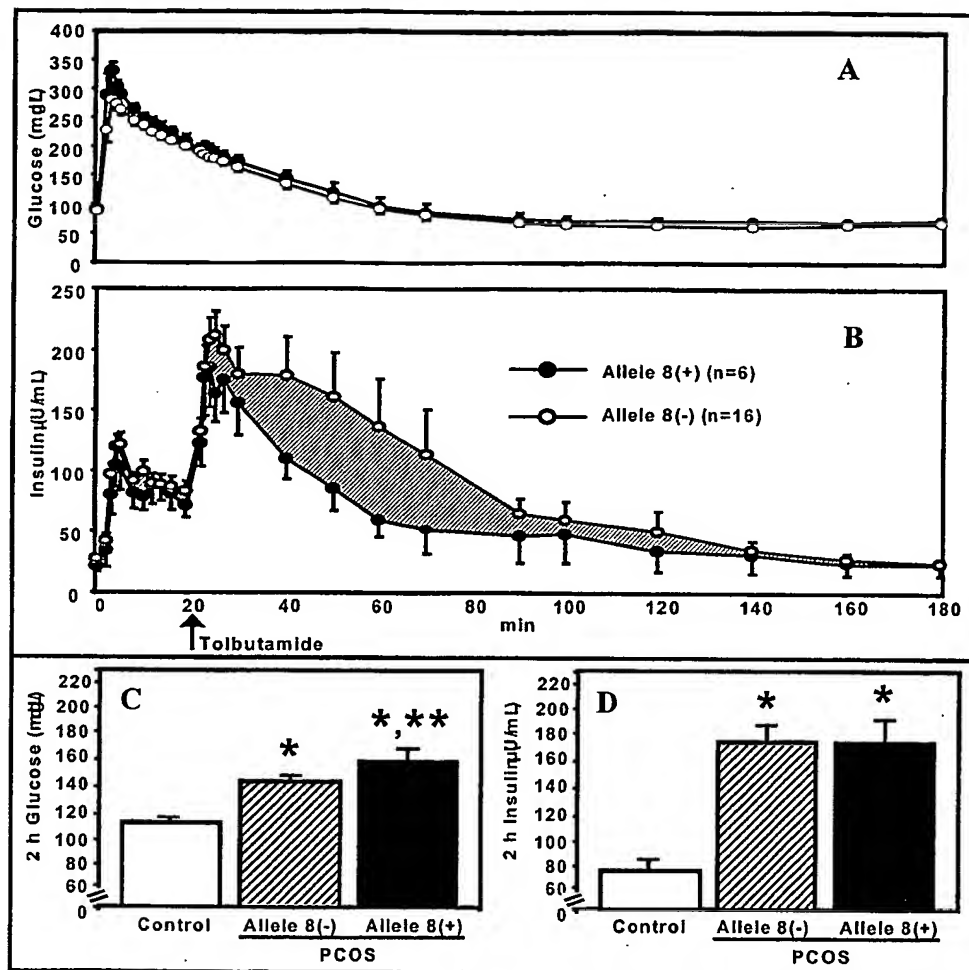


Figure 3. FSIGT glucose (A) and insulin (B) responses and 2 h post-75g glucose (C) and insulin (D) levels in obese A8(+) and A8(-) PCOS women. Tolbutamide, 500mg iv, given at 20 min of the FSIGT. The shaded area in panel B is the difference in insulin responses in A8(+) vs A8(-) PCOS. * $P < 0.05$ vs weight matched control women, ** $P < 0.05$ vs A8(-) PCOS, by ANCOVA adjusted for age.

FIGURE NO. 4

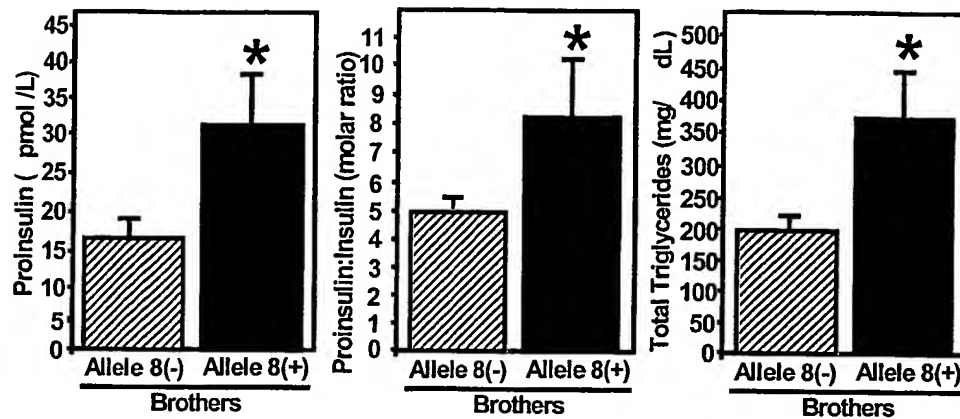


Figure 4. Fasting proinsulin, proinsulin:insulin and total triglyceride levels in obese A8(-) and A8(+) brothers of PCOS women, *P<0.05.

FIGURE NO. 5

Hypothesis for prenatal androgen programming of females for PCOS

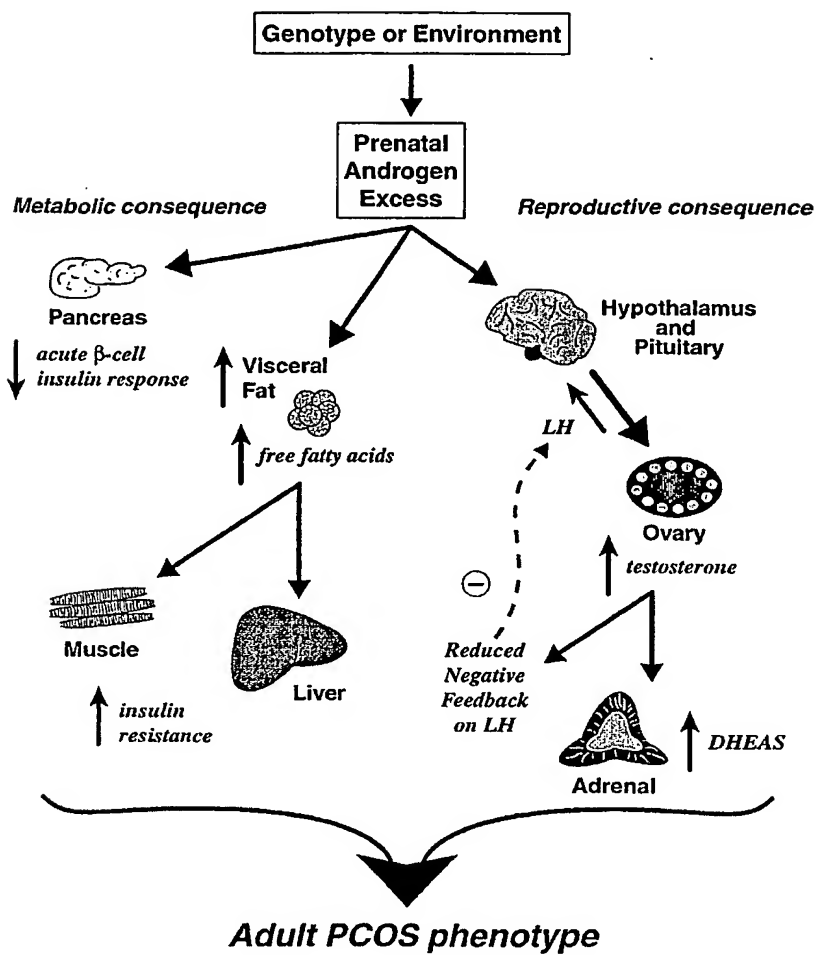


Figure 5. Hypothesis for genetic variation resulting in androgen excess, which causes metabolic and reproductive defects by prenatal programming.